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Conceptual Paper

Hantavirus Infection: A Possible Zoonosis!

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Abstract

Hantavirus pulmonary syndrome (HPS) is a severe infectious disease characterized by pulmonary oedema followed by respiratory disorder and shock. Hantaviruses infects the lung endothelium without causing any cytopathic effect. However, virus causes mircovascular leakage, which is the hallmark of HPS. The HPS associated viruses are member of genus Hantavirus and family Bunyaviridae. Hantaviruses worldwide are broadly split into the new world Hantaviruses, which includes those causing HPS, and the old-world Hantaviruses (including the prototype Hantaan virus [HTNV]), which are associated with hemorrhagic fever with renal syndrome (HFRS). Sin Nombre virus (SNV) and Andes virus (ANDV) are the most common causes of HPS. Case fatality of HPS is approximately 40%.

Keywords: Hantavirus; Shock; Bunyaviridae; Hemorrhagic Fever; Sin Nombre Virus

Introduction

Orthohantaviruses are emerging rodent-borne pathogens which cause Hantavirus Pulmonary Syndrome in humans. Hantavirus was named after Hantan river area in South Korea. It was isolated from the lungs of striped field mouse, *Apodemus agrarius.* The first outbreak occurred during Korean War (1950 - 1953) in which more than 3000 United Nations troops developed hemorrhagic fever along with renal syndrome (HFRS), also referred to as old world Hantavirus. This outbreak sparked a 25-year search for the causative agent. The second outbreak of disease occurred in United States (1993) which is called as Hantavirus Pulmonary Syndrome (HPS) or 'new world Hantavirus'. The virus responsible for 1993 cases in America is now called as Sin Nombre virus which is responsible for Hantavirus Cardiopulmonary syndrome (HCPS or HPS).

Etiology

Genus Orthohantavirus is a single-stranded, enveloped, negative- sense segmented RNA viruses within Bunyaviridae family. Hantavirus is a spherical enveloped and medium-sized i.e., 95-120 nm RNA virus which has tessellated appearance under electron microscope. There are three genomic segments which are defined as S (small), M (medium), L (large). These genomic segments namely S, M and L are responsible for encoding the nucleoprotein, enveloping glycoprotein (Gn and Gc) and functioning of RNA dependent RNA polymerase respectively. Of more than 20 known species of rodent borne viruses of genus Hantavirus, 11 cause human disease [3]. There are two major forms of disease: the hemorrhagic fever with renal syndrome (HFRS) and Hantavirus cardiopulmonary syndrome (HCPS or HPS). The most severe forms of HPS agents are associated with Sin Nombre virus (SNV) and the southern (proto-

typical) form of Andes virus; slightly milder forms caused by the northern form of Andes virus, Laguna Negra virus (LNV), and Choclo virus [2]. Generally, case-fatality ratios of Hantavirus pulmonary syndrome ranges from 30 to 50% for milder forms.

Transmission

Orthohantavirus are only virus of family *Bunyaviridae* that are not arthropod borne. The hallmark of virus infection in rodent reservoir hosts is persistent, usually life-long and inapparent infection shedding in urine, saliva and faeces. Human diseases are caused by contact with contaminated feed and water by rodent excreta or saliva, usually in winters. Andes Orthohantavirus is the only confirmed Hantavirus capable of causing transmission from person to person, although this is rare. Human infection after rodent bite has also been reported. People who work as animal trappers, forestry workers, farmers, etc. are at higher risk.

Rodents that carry Hantavirus are

- Cotton rat (*Sigmodon hispidus)*
- Deer mouse (*Permyscus maniculatus)*
- Rice rat (*Oryzomys palustris)*
- White footed mouse (*Permyscus leucopus)*

Pathogenicity

Hantavirus infection causes a non- pathogenic, persistent infections in rodents. In humans, it is characterized by alterations in endothelial cells permeability and vascular oedema and cause hemorrhagic fever with renal syndrome (HFRS) and pulmonary syndrome, both with high mortality. Beta 3 integrin's mediate entry of pathogenic hantaviruses. Increased capillary permeability is primarily an important factor in the pathogenesis of hantavirus infections [1]. Inflammatory mediators of the host immune response plays an important role in the capillary leak that may result into sudden hypotension and shock. When virus enters the body, it first crosses the mucus gel that covers the respiratory endothelial cells. After crossing it, the virus crosses the respiratory epithelium. It is not fully clear how hantavirus passes through respiratory epithelium as the epithelium has tight junction integrity and forms a particle impermeable barrier. There is bidirectional spread of virus from apical membrane and basolateral membrane and the virus spreads into lung endothelium and distant parts of the body through dendritic cells and activated macrophages. Virus can be found in numerous capillaries and blood vessels of various organs. Virus causing HFRS (Haemorrhagic fever with renal syndrome) has affinity for podocytes in the glomerulus and tubular epithelial cells of kidney.

Clinical signs (Gross)

Hantavirus is a life-threatening disease. The disease first begins with high fever, chills, headache, back pain, abdominal pain along with nausea and vomiting. Based on geographical distribution of Hantavirus i.e., Hantavirus pulmonary syndrome and Hemorrhagic fever along with renal syndrome; are the two clinical manifestations. Other sign involves dry cough, chest pain, low blood pressure, dizziness, renal failure etc. [4].

Other changes

Hantavirus is mainly characterized by pulmonary oedema, hypoxia, and hypotension. Major changes are observed are in lungs, kidneys. Thrombocytopenia, leukocytosis, hemoconcentration, hypoalbuminaemia, and increase in LDH can also be observed. Simultaneous appearance of thrombocytopenia and an immunoblast count exceeding 10% of total lymphoid series can be used for diagnostic triad.

Diagnosis

- **Serological test:** All HPS and HFRS cases have high IgM and IgG antibodies ratio to N protein. Hence serological tests that detect IgG and IgM antibodies to hantavirus antigens in serum are the most common techniques for the diagnosis of suspected cases of HPS and HFRS. Hantavirus antigens which are currently used for serological tests are mostly derived by using recombinant DNA methods.
- **Molecular diagnosis:** Within 12 to 24 hours, a patient can evolve from acute feverish illness to severe pneumonia along with cardiogenic shock and respiratory failure. Therefore, it is necessary to have a rapid diagnostic test for patients. Genome of hantavirus can be rapidly diagnosed by reverse transcription PCR with clinical samples such as blood, serum or organ fragments from the day one after onset of the illness [5].

Conclusion

Hantavirus is carried by various rodent species throughout the world. Towards the end of 2019-2020s, a wave of coronavirus disease (COVID-19) pandemic has started the discussion about the other possible communicable infectious diseases. Hantavirus is a significant zoonotic pathogen and common initial symptoms of HPS are similar to those of COVID-19. Respiratory disorders arising as a result of hanta virus can proceed rapidly to respiratory failure, resulting from pulmonary oedema and shock. This could lead to

more number of mortality as compared to COVID-19 infection. Till date, there is no effective treatment and vaccines available for this disease so public awareness and precautionary measurements can help reducing the spread of Hantavirus.

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